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Repeated respiratory hospital encounters among children with asthma and residential proximity to traffic

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ABSTRACT

Objective: The prevalence of adverse respiratory outcomes among children has been frequently associated with measurements of traffic-related exposures, and other data suggest asthma severity is worsened with residence near heavy traffic. We examined the association between neighbourhood traffic burden and repeated acute respiratory illnesses that required emergency department visits and/or hospitalisation for children with a primary or secondary diagnosis of asthma (89% acute bronchitis or pneumonia).

Methods: This is a hospital-based longitudinal study of a southern California urban catchment area around two adjacent children's hospitals. Subjects' home addresses were geocoded and linked to nearby traffic data. Recurrent event proportional hazard analysis was used to estimate the hazard of repeated hospital encounters.

Results: We found living within 300 metres of arterial roads or freeways increased risk of repeated hospital encounters in 3297 children age 18 years or less. At highest risk were children in the top quintile of traffic density (HR = 1.21; 95% CL 0.99 to 1.49) and those who had 750 metres or more of arterial road and freeway length within 300 metres of their residence (HR = 1.18; 95% CL 0.99 to 1.41). Associations between repeated hospital encounters and residence near heavy traffic were stronger in females than males and in children without insurance or who required government sponsored insurance than children with private insurance. The gender disparity was most notable among infants (age 0) and children ages 6–18 years.

Conclusions: Results suggest exposure to traffic-related air pollution increases asthma severity as indicated by hospital utilisation. The finding in infants suggests this is an especially vulnerable population, although the validity of asthma diagnosis at this age is unknown. Females and children who do not have private insurance may also be more vulnerable to air pollution from traffic.

Epidemiological studies of paediatric asthma and air pollution have focused on the impacts of air pollutants measured at regional air monitoring stations. Although this has contributed valuable insight to the impact of regulated criteria air pollutants such as ozone and airborne particle mass, the health impacts of unmonitored toxic pollutants near their sources may be greater. Traffic emissions are a major source of neighbourhood air pollution and various measurements of traffic-related exposures have been consistently linked to adverse respiratory health effects among children.^{1–9} Many of these epidemiological studies suggest that traffic-related air pollution can exacerbate and possibly

induce the initial expression of asthma. Recent published cohort studies in southern California found that children who live within 500 metres of a freeway have deficits in lung function development,³ while children who reside within 75 metres of a major road are at an increased risk of being diagnosed with asthma.⁴

There are some data showing positive associations between asthma or wheezing severity in children and residence near traffic, including increased risk of emergency department (ED) visits or hospital encounters,^{6, 10} and increased asthma symptom severity or wheezing.^{8, 11–13} In addition, a few studies have examined the potential part that gender plays in modifying the relation between traffic-related air pollution and adverse respiratory outcomes in children with asthma.^{4, 5, 7, 9}

We investigated the risk of repeated hospital encounters (hospitalisation or ED visits) from residential exposure to traffic using a retrospective hospital-based cohort study. We also examined potential differences in association by insurance status, age group and gender. We developed traffic exposure metrics to estimate local traffic-related air pollution exposure levels at the homes of children with a primary or secondary diagnosis of asthma.

METHODS

Study population

Subjects for the study were drawn from hospital admission and discharge records from the Children's Hospital of Orange County (CHOC) and the University Children's Hospital of the University of California Irvine Medical Center (UCIMC) for the period between 1 January 2000 and 31 December 2003. The inclusion criteria were as follows: aged 18 years or younger; one or more respiratory hospital encounters for a primary or secondary diagnosis of asthma (ICD-9 493) within the study period; and home residence in census block areas located within 13 km of either UCIMC or CHOC (catchment area). The two hospitals are located within 2.5 km of each other. We selected hospital encounters for a secondary asthma diagnosis because these encounters were probably the result of the impacts of the primary diagnosis on asthma symptoms. Most of the primary diagnoses in these cases were for acute bronchitis (19.5%) or pneumonia (69.1%). A unique hospital encounter for the present study was defined as either a visit to the ED or hospitalisation that occurred at least eight days apart. The 13 km radius from the two hospitals was chosen based on GIS density

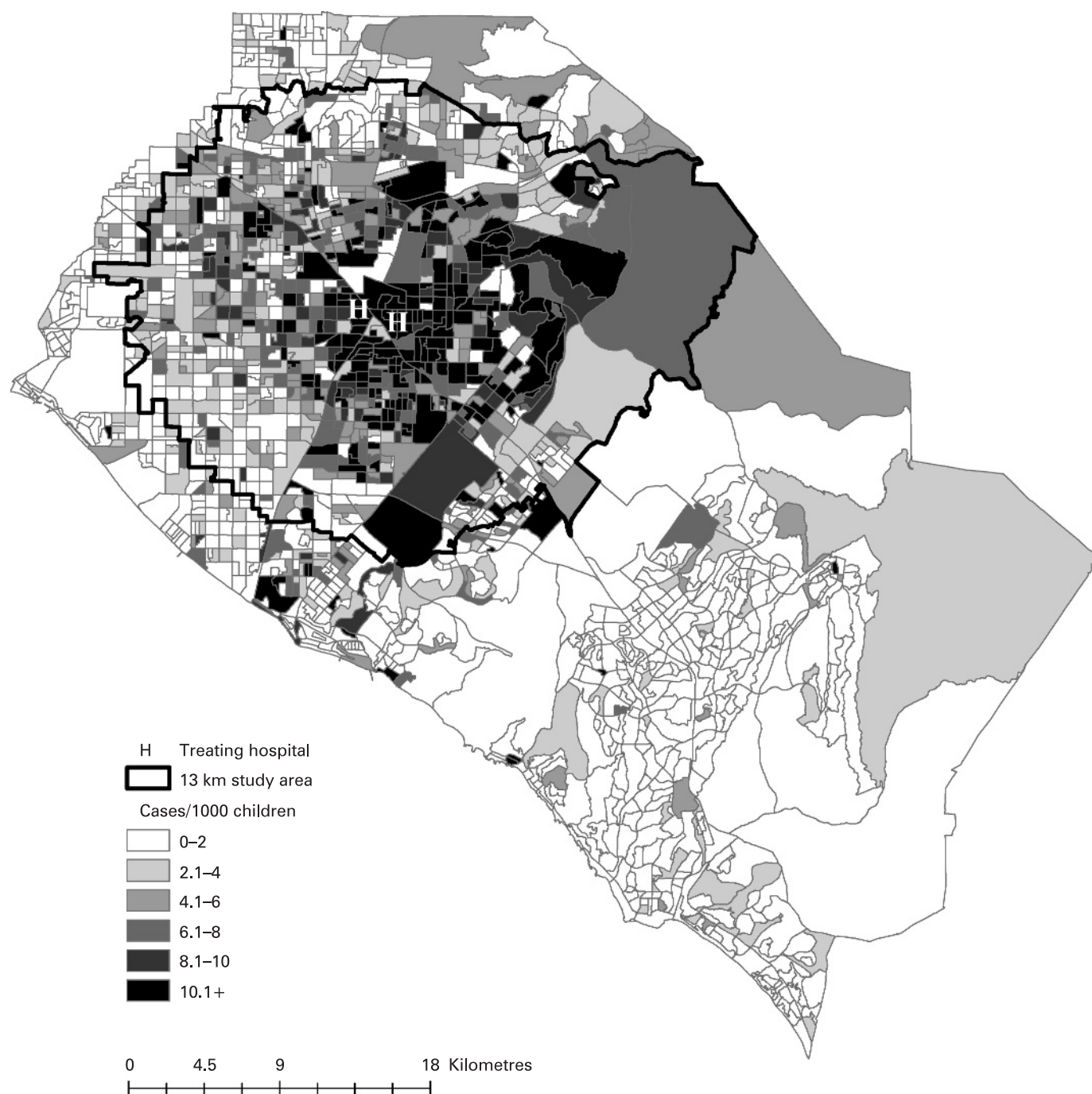


Figure 1 Children diagnosed with primary or secondary asthma admitted to University of California, Irvine Medical Center and Children's Hospital of Orange County, 2000-3 by census block group.

mapping data, which revealed that the majority of children who utilised UCIMC or CHOC resided within 13 km of the hospitals (fig 1). Distance from residence to hospital was associated with the odds of return, but not if the child resided within the 13 km catchment area.

Exposure assessment

EZ-Locate (Tele Atlas North America Inc, Boston, MA, USA) was used to geocode residential addresses reported at the first hospital encounter. We succeeded in obtaining an exact address match for 3712 of 3983 children in the database (93%). Of the 3712 children, 3297 resided within the 13-km catchment area.

ArcView GIS was used to calculate three traffic proxies reflecting local traffic-related air pollution exposure levels. For the first traffic metric, we calculated the shortest distance from each child's primary residence to the nearest major road (arterial road or freeway). Thereafter, a 300-metre buffer was drawn around each child's residence to reflect an "exposure zone" to local traffic-related air pollution. The buffer distance selection was based on studies by Zhu *et al*¹⁴ and Hitchins *et al*,¹⁵ which found a significant decrease in the concentration of ultrafine particles (<0.1 µm in diameter), black carbon and CO to background concentrations by 300 metres. We consider these pollutant measurements to be markers of primary combustion aerosols from traffic, including diesel exhaust rich in redox

active components believed to be common exposures involved in asthma exacerbations.¹⁶

We then calculated the total length of major roads within the 300-metre buffer by summing up all arterial road and/or freeway lengths within the 300-metre buffer. Lastly, we calculated neighbourhood traffic density by dividing the total vehicle metres travelled (VMT) within the 300-metre buffer by the area of the buffer. VMT was calculated by multiplying the length of major road segments within the 300-metre buffer by their associated average daily traffic (ADT) count obtained from the Orange County Transportation Authority (OCTA). The area of the buffer was calculated by multiplying π and the buffer radius squared. Traffic density was computed as

$$\text{Traffic density} = \frac{\text{VMT}_{\text{Total}}}{(300)^2} \text{ (VMT/day/m}^2\text{)}$$

Statistical analysis

Recurrent event proportional hazards analysis was used to estimate the effect of traffic-related air pollution on hospital readmissions among children with asthma. Patients were considered at risk for an event from the time of first hospital encounter until their 19th birthday or the end of the observation period, whichever occurred first. The primary outcome of interest in the analysis was the combined endpoint of ED visit and hospital admission. In the recurrent events analysis, we allowed for separate baseline hazards at each recurrence level and estimated the pooled log-hazard ratio associated with each adjustment covariate over all recurrence levels.¹⁷ We considered up to 10 readmissions per subject in the analysis, as nearly all subjects experienced 10 or fewer readmissions over the course of follow-up. We adjusted our models with known risk factors that may have confounded the relation between traffic burden and repeated hospital encounters. The risk factors adjusted for include gender, age group, race, insurance status, residence distance to treating hospital and census block group median household income. Robust variance estimates were used for all inferences in order to account for within-subject correlation caused by the recurrent event nature of the data.¹⁸ The proportional hazards assumption was assessed by considering covariate by time interactions. No significant deviations from the proportional hazards assumption were observed.

The biological predisposition of children to the effects of traffic-related air pollution exposure probably varies with age, gender and social economic status (SES).^{4 5 7 9 19} To examine these differences, subgroup analyses were performed by age group (0, 1–5, 6–18 years). Asthma is more clearly diagnosed in school children than in preschool ages when wheezing from respiratory infections is common. This is in part a justification for the use of a secondary diagnosis of asthma since we are also interested in the impact of traffic-related air pollution on preschool children who wheeze. To examine the potential effect modification between gender and traffic exposure, and between insurance status and traffic exposure, interaction terms were added to the model. Estimated hazard ratios corresponding to subgroups derived from interaction terms were obtained by exponentiating the appropriate parameter estimate contrast, and associated Wald-based confidence limits and p values were calculated. Global tests for interaction terms were conducted using the partial likelihood ratio test. Statistical analysis was completed in SAS V8.2 and R V2.4.1.

RESULTS

A total of 3297 children representing 4760 hospital encounters were identified as meeting the study criteria, of which 2480 children (75%) had one hospital encounter during the study period and 817 children (25%) had two or more. Among the children who had repeated hospital encounters during the study period, 499 (61%) had two, 163 (20%) had three, 79 (10%) had four and 42 (5%) had five hospital encounters. Only 34 (4%) of the children had six or more hospital encounters during the study period. Hospital encounters for a primary diagnosis of asthma numbered 4018 (84%) and 742 were for a secondary diagnosis of asthma (16%).

Table 1 shows the descriptive statistics for all children in the study by demographics and hospital encounter readmission status. For children with readmissions, 78% of the first readmissions occurred within one year from the initial study enrolment date. Over half of the children in the study were between the ages of 0 to 5 years, including 69% of those with readmissions. The majority of subjects were non-Hispanic white or white Hispanic, and resided within 10 km of their respective treating hospital. Fifty-nine per cent of the study population resided in census block group areas where the median household income was below \$50 000 and approximately half of the study population paid for their medical visit through government-sponsored health insurance or self payment. The study gender distribution consisted of more boys than girls (58% boys vs 42% girls) reflecting the typical male predominance of asthma until adolescence.

Table 2 displays the estimated risk of repeated hospital encounters given exposure to three estimated traffic-related air pollution metrics (each modelled separately) for children aged 18 years or younger with asthma. The results indicate that children diagnosed with asthma with 750 metres or more of major road length within 300 metres of their residences experienced a rate of repeated hospital encounters that was 18% higher than children without major roads within 300 metres (HR = 1.18; 95% CL 0.99 to 1.41). Children experienced higher rates of repeated hospital encounters if the distance to major roads was within 300 metres of their residences. There was no evidence of a dose-response relation for distances closer than 150–300 metres. A potential dose-response relation was suggested for traffic density and the hazard of repeated hospital encounters. Children residing in the fifth quintile of traffic density (113 VMT/day/m²) had a repeated hospital encounter rate that was 21% higher than those children without major roads within 300 metres of their residences (HR = 1.21; 95% CL 0.99 to 1.49). We did not observe any difference in the association with traffic density if they resided more or less than 150 metres from a major road. p Values for trend for total major road length (p = 0.45), distance to nearest major road (p = 0.54) and traffic density (p = 0.66) were not statistically significant. Traffic effects were observed to be stronger among those with multiple hospital encounters. However, owing to sample size limitations we did not observe consistent increases in risk across each additional encounter.

We examined the potential modifying effect that gender and insurance status (proxy for SES) may have on the relation between traffic-related air pollution exposure and repeated hospital encounters for children 18 years and younger. We found the effect of traffic exposure to be stronger in females and among children with no insurance or government-sponsored insurance. Females and children with government-sponsored insurance or no insurance residing in the top fifth quintile of traffic density had a rate of repeated hospital encounters that

Table 1 Demographic characteristics by hospital encounter status (n = 3297 children)

	Readmission	
	No (n) (%)*	Yes (n) (%)*
Gender		
Female	1070 (43.15)	327 (40.02)
Male	1410 (56.85)	490 (59.98)
Age group at study entry (years)		
0	380 (15.32)	236 (28.89)
1–5	1082 (43.63)	327 (40.02)
6–13	806 (32.50)	223 (27.29)
14–18	212 (8.55)	31 (3.79)
Race		
White non-Hispanic	1072 (43.23)	392 (47.98)
White Hispanic	1087 (43.83)	360 (44.06)
Black	66 (2.66)	17 (2.08)
Asian	69 (2.78)	18 (2.20)
Other	124 (5.00)	21 (2.57)
Unknown	62 (2.50)	9 (1.10)
Insurance status at study entry		
Private	921 (37.14)	344 (42.11)
Government-sponsored or self-pay	1239 (49.96)	425 (52.02)
Unknown	320 (12.90)	48 (5.88)
Resident distance to treating hospital		
0–5 km	748 (30.16)	274 (33.54)
6–10 km	1211 (48.83)	371 (45.41)
11+ km	521 (21.01)	172 (21.05)
Census block based median household income		
≤ \$29 999	180 (7.26)	64 (7.83)
\$30 000–\$39 999	687 (27.7)	226 (27.66)
\$40 000–\$49 999	586 (23.63)	203 (24.85)
\$50 000–\$59 999	441 (17.78)	150 (18.36)
\$60 000 +	586 (23.63)	174 (21.30)
Year of study enrolment		
2000	571 (23.02)	405 (49.57)
2001	630 (25.40)	234 (28.64)
2002	577 (23.27)	102 (12.48)
2003	702 (28.31)	76 (9.30)
Time to first readmission		
1–2 months	–	217 (26.56)
3–6 months	–	242 (29.62)
7–12 months	–	177 (21.66)
After 1 year	–	181 (22.15)

*Column percentage.

was almost 50% higher than those children without major roads within 300 metres. For males and children with private insurance, no statistically significant increase was found in the rate of repeated hospital encounters at any level of traffic exposure within 300 metres of their residences. The interaction between insurance status and traffic exposure was statistically significant for residence distance to nearest major road ($p = 0.01$), and nearly significant for total major road length ($p = 0.06$) and traffic density ($p = 0.16$). However, tests for interaction between gender and traffic exposure level did not reach statistical significance for any of the traffic metrics.

We then examined the potential modifying effect that gender may have on the relation between traffic-related air pollution exposure and repeated hospital encounters for children at different ages. We tested interaction terms of gender by exposure for each age group (table 3). Overall, the association between the risk of hospital encounters and traffic indexes for subjects residing near heavy traffic was greater in female infants

than in male infants diagnosed with asthma (table 3). For male infants, no significant increase was found in the rate of repeated hospital encounters at any level of traffic exposure within 300 metres of their residences. However, tests for the interaction between gender and traffic exposure level did not reach statistical significance for any of the traffic metrics.

Tables 4 and 5 show the risk of repeated hospital encounters given estimated traffic-related air pollution exposure for children aged 1–5 and 6–18 by gender. No significant increase in rate of repeated hospital encounters was observed for the three traffic proxies for either gender in the 1–5 year old age group. However, we observed that both girls and boys age 6–18 years with major roads within 300 metres of their residences experienced higher rates of repeated hospital encounters than girls or boys without major roads within 300 metres of their residences. Although the increase in risk observed in girls was statistically significant at many traffic exposure levels, only non-significant increased HRs were observed for boys in this age group. However, a test for the interaction between gender and traffic exposure level was not statistically significant for the 6–18-year-old age group.

DISCUSSION

In this hospital-based longitudinal study, we found an association between living within 300 metres of major roads and increased risk of repeated hospital encounters in children aged 18 years or younger with asthma. We found some evidence of a dose-response association for traffic density and total major road length within 300 metres of residence. However, some categories in several models were inconsistent with a dose-response relation. For residence distance to nearest major road, the largest effect we observed was for the category of 150–300 metres. The lack of a dose-response relation may be partly attributable to the variation in traffic intensity experienced at each distance level depending on the type of major road (arterial or freeway) and its associated ADT count.

Findings suggest that neighbourhood traffic-related air pollution can lead to a respiratory illness severe enough to require an ED visit or hospital admission in children with a primary or secondary diagnosis of asthma at the hospital encounter. Our findings are coherent with recent studies that link neighbourhood traffic to increased asthma prevalence and reduced lung function in schoolchildren living in southern California where traffic is the predominant source of air pollution.^{3,4} Children are more susceptible to respiratory illnesses because they have immature lungs and immune systems. This is especially true of infants. Children also tend to spend more time outdoors breathing at higher respiratory rates and inhaling more air per unit of body mass than adults.¹ Particulate matter (PM) air pollution released from vehicle exhaust and inhaled by children has been suggested to cause biological damage at three different levels. First, inhaled PM can cause oxidative stress in macrophage and epithelial cells resulting in airway structural damage. Second, ultrafine PM <100 nm in diameter can directly damage mitochondria structurally, impair ATP production and induce apoptosis in macrophages and epithelial cells. Third, PM has the potential to trigger an immunological cascade response to oxidative stress when antioxidant responses are overwhelmed. These pathways can lead to an adverse respiratory event in predisposed children with asthma,^{20,21} particularly given evidence for reduced antioxidant enzyme capacity in the lungs and peripheral blood²² of patients with asthma. Environmental studies have also found that particle concentrations from traffic-related air pollutants (ultrafine PM, black carbon and

Table 2 Residential traffic exposure as a predictor for the hazard of repeated hospital encounters for children age 0 to 18 years diagnosed with asthma†‡

Traffic index	Events/ censored	By insurance status§			By gender	
		All subjects	Private	Government/self pay	Boys	Girls
		HR (95% CL)	HR (95% CL)	HR (95% CL)	HR (95% CL)	HR (95% CL)
Total arterial road and freeway length within 300 metres of residence				p = 0.06**		p = 0.33††
No arterial road or freeway within 300 metres	143/366	1.00	1.00	1.00	1.00	1.00
<750	651/1469	1.13 (0.95 to 1.34)	0.91 (0.71 to 1.17)	1.43 (1.11 to 1.84)*	1.08 (0.87 to 1.34)	1.24 (0.92 to 1.67)
≥750	668/1459	1.18 (0.99 to 1.41)**	1.03 (0.81 to 1.32)	1.48 (1.15 to 1.90)*	1.08 (0.87 to 1.34)	1.39 (1.04 to 1.87)*
Residence distance (metres) to nearest arterial road or freeway				p = 0.01**		p = 0.48††
>300	143/366	1.00	1.00	1.00	1.00	1.00
150–300	471/1003	1.21 (1.00 to 1.45)*	0.92 (0.71 to 1.20)	1.54 (1.19, 2.00)*	1.12 (0.88 to 1.41)	1.40 (1.03 to 1.89)*
50–150	461/1028	1.14 (0.95 to 1.37)	1.04 (0.80 to 1.34)	1.36 (1.05 to 1.77)*	1.10 (0.88 to 1.38)	1.23 (0.90 to 1.68)
<50	387/897	1.11 (0.92 to 1.33)	0.93 (0.72 to 1.22)	1.45 (1.12 to 1.88)*	1.00 (0.80 to 1.25)	1.31 (0.96 to 1.80)
Traffic density (VMT/day/m^{††}) within 300 metres of residence¶				p = 0.16**		p = 0.58††
No arterial road or freeway within 300 metres	143/366	1.00	1.00	1.00	1.00	1.00
1st quintile (<27 VMT/day/m ²)	257/590	1.13 (0.92 to 1.38)	0.87 (0.65 to 1.16)	1.47 (1.10 to 1.97)*	1.04 (0.80 to 1.33)	1.31 (0.94 to 1.84)
2nd quintile (28–47)	256/591	1.11 (0.90 to 1.35)	0.80 (0.59 to 1.09)	1.51 (1.15 to 2.00)*	1.02 (0.79 to 1.32)	1.28 (0.91 to 1.80)
3rd quintile (48–71)	263/584	1.15 (0.94 to 1.40)	1.08 (0.81 to 1.43)	1.31 (0.99 to 1.74)**	1.10 (0.86 to 1.41)	1.26 (0.90 to 1.75)
4th quintile (72–112)	267/579	1.19 (0.98 to 1.45)	1.04 (0.79 to 1.36)	1.48 (1.12 to 1.97)*	1.17 (0.92 to 1.49)	1.25 (0.90 to 1.74)
5th quintile (>113)	274/576	1.21 (0.99 to 1.49)**	1.08 (0.80 to 1.45)	1.48 (1.12 to 1.96)*	1.07 (0.82 to 1.38)	1.49 (1.08 to 2.05)*

†Adjusted for race, age group, gender, insurance status, residence distance to treating hospital and median household income.

‡Hazard ratio, confidence limit and p value from recurrent event proportional hazard model.

¶Excluded observations where an arterial road is present within 300 metres of residence but no data are available for ADT.

§Insurance status was unknown for 368 children (11%), making interpretation of the null results difficult for this small group (not shown in table 2).

**Global test for interaction between insurance status and traffic metrics. p Value from partial likelihood ratio test.

††Global test for interaction between gender and traffic metrics. p Value from partial likelihood ratio test.

*p ≤ 0.05; **p ≤ 0.06.

VMT, total vehicle metres travelled.

Table 3 Residential traffic exposure as a predictor for the hazard of repeated hospital encounters for children with asthma by age group at study entry and gender: age 0†‡

Traffic index	Boys		Girls		p Value§
	Events/censored	HR (95% CL)	Events/censored	HR (95% CL)	
Total arterial road and freeway length within 300 metres of residence					0.11
No arterial road or freeway within 300 metres	43/42	1.00	12/25	1.00	
<750	154/155	0.97 (0.68 to 1.38)	70/119	1.21 (0.67 to 2.14)	
≥750	134/162	0.89 (0.61 to 1.30)	94/112	1.57 (0.90 to 2.75)	
Residence distance to nearest arterial road or freeway					0.69
>300	43/42	1.00	12/25	1.00	
150–300	97/105	0.96 (0.66 to 1.40)	56/75	1.39 (0.77 to 2.49)	
50–150	96/115	0.88 (0.59 to 1.31)	51/82	1.28 (0.71 to 2.31)	
<50	95/97	0.96 (0.67 to 1.38)	57/74	1.49 (0.82 to 2.68)	
Traffic density within 300 metres of residence¶					0.11
No arterial road or freeway within 300 metres	43/42	1.00	12/25	1.00	
1st quintile (<27 VMT/day/m ²)	51/59	0.96 (0.61 to 1.51)	31/39	1.57 (0.84 to 2.95)	
2nd quintile (28–47)	79/76	1.01 (0.69 to 1.49)	31/47	1.31 (0.69 to 2.49)	
3rd quintile (48–71)	53/72	0.84 (0.55 to 1.30)	33/47	1.31 (0.70 to 2.48)	
4th quintile (72–112)	72/52	1.14 (0.77 to 1.69)	27/46	1.24 (0.65 to 2.38)	
5th quintile (>113)	32/57	0.63 (0.38 to 1.03)	42/50	1.58 (0.87 to 2.89)	

†Adjusted for race, insurance status, residence distance to treating hospital and median household income.

‡Hazard ratio, confidence limit and p value from recurrent event proportional hazard model.

¶Excluded observations where an arterial road is present within 300 metres of residence but no data are available for ADT.

§Global test for interaction between gender and traffic metrics. p Value from partial likelihood ratio test.

*p ≤ 0.05.

VMT, total vehicle metres travelled.

Table 4 Residential traffic exposure as a predictor for the hazard of repeated hospital encounters for children with asthma by age group at study entry and gender: age 1–5†‡

Traffic index	Boys		Girls		p Value§
	Events/censored	HR (95% CL)	Events/censored	HR (95% CL)	
Total arterial road and freeway length within 300 metres of residence					0.75
No arterial road or freeway within 300 metres	36/98	1.00	23/58	1.00	
<750	150/375	1.05 (0.75 to 1.47)	104/266	1.11 (0.73 to 1.69)	
≥750	156/359	1.13 (0.81 to 1.58)	93/251	1.04 (0.69 to 1.56)	
Residence distance to nearest arterial road or freeway					0.82
>300 metres	36/98	1.00	23/58	1.00	
150–300	108/259	1.10 (0.77 to 1.56)	73/174	1.13 (0.73 to 1.75)	
50–150	130/279	1.16 (0.83 to 1.64)	58/168	1.02 (0.65 to 1.60)	
<50	68/196	0.96 (0.67 to 1.39)	66/175	1.06 (0.69 to 1.62)	
Traffic density within 300 metres of residence¶					0.65
No arterial road or freeway within 300 metres	36/98	1.00	23/58	1.00	
1st quintile (<27 VMT/day/m ²)	54/147	0.97 (0.66 to 1.45)	27/96	0.85 (0.49 to 1.48)	
2nd quintile (28–47)	54/144	1.00 (0.66 to 1.52)	49/108	1.31 (0.82 to 2.11)	
3rd quintile (48–71)	70/161	1.17 (0.81 to 1.69)	45/96	1.21 (0.76 to 1.92)	
4th quintile (72–112)	58/136	1.10 (0.73 to 1.66)	30/111	0.87 (0.54 to 1.41)	
5th quintile (>113)	69/142	1.21 (0.83 to 1.77)	46/106	1.11 (0.71 to 1.74)	

†Adjusted for race, insurance status, residence distance to treating hospital and median household income.

‡Hazard ratio, confidence limit and p-value from recurrent event proportional hazard model.

¶Excluded observations where an arterial road is present within 300 metres of residence but no data is available for ADT.

§Global test for interaction between gender and traffic metrics. p Value from partial likelihood ratio test.

*p ≤ 0.05.

VMT, total vehicle metres travelled.

CO) generally dissipate by half at around 150 metres and decline to background concentrations by 300 metres from the upwind source road during the day,¹⁴ but not at night.²⁵ This means that children who live closest to roads with high traffic density are exposed to the most traffic-related air pollution.

However, our findings suggest that exposures within 150–300 metres are important.

Insurance status was found to modify the effect of traffic exposure on repeated hospital encounters. Associations were notably stronger among children without insurance or who

Table 5 Residential traffic exposure as a predictor for the hazard of repeated hospital encounters for children with asthma by age group at study entry and gender: age 6–18†‡

Traffic index	Boys		Girls		p Value§
	Events/censored	HR (95% CL)	Events/censored	HR (95% CL)	
Total arterial road and freeway length within 300 metres of residence					0.43
No arterial road or freeway within 300 metres	19/80	1.00	10/63	1.00	
<750	100/313	1.20 (0.78 to 1.86)	73/241	1.53 (0.83 to 2.84)	
≥750	97/314	1.21 (0.79 to 1.86)	94/261	1.90 (1.03 to 3.52)*	
Residence distance to nearest arterial road or freeway					0.70
>300	19/80	1.00	10/63	1.00	
150–300	70/219	1.25 (0.79 to 1.98)	67/171	1.96 (1.06 to 3.64)*	
50–150	75/214	1.31 (0.84 to 2.05)	51/170	1.66 (0.87 to 3.19)	
<50	52/194	1.03 (0.66 to 1.62)	49/161	1.46 (0.75 to 2.83)	
Traffic density within 300 metres of residence¶					0.67
No arterial road or freeway within 300 metres	19/80	1.00	10/63	1.00	
1st quintile (<27 VMT/day/m ²)	53/151	1.28 (0.78 to 2.09)	41/98	1.87 (0.97 to 3.60)	
2nd quintile (28–47)	23/122	0.80 (0.46 to 1.38)	20/94	1.22 (0.60 to 2.48)	
3rd quintile (48–71)	37/103	1.37 (0.83 to 2.26)	25/105	1.39 (0.67 to 2.89)	
4th quintile (72–112)	41/124	1.27 (0.78 to 2.05)	39/110	2.10 (1.08 to 4.06)*	
5th quintile (>113)	43/127	1.34 (0.83 to 2.18)	42/94	2.10 (1.06 to 4.15)*	

†Adjusted for race, insurance status, residence distance to treating hospital and median household income.

‡Hazard ratio, confidence limit and p-value from recurrent event proportional hazard model.

¶Excluded observations where an arterial road is present within 300 m of residence but no data is available for ADT.

§Global test for interaction between gender and traffic metrics. p Value from partial likelihood ratio test.

*p ≤ 0.05.

VMT, total vehicle metres travelled.

required government-sponsored insurance. Children without insurance or utilised government insurance can generally be inferred to be from lower SES households. It has been suggested that lower SES children may be more susceptible to pollutants given that they may have enhanced responses as a result of other factors more common in this group, including allergens and psychosocial stress.¹⁹ Children in families without health insurance may also have inadequate preventive care, which is critical in controlling asthma flares.

We also found evidence for a modifying effect of age and gender. Associations were particularly strong for females and infants diagnosed with asthma. Around half of the repeat encounters among infants occurred between the ages of 1–3 years. Could early life responses to traffic exposures have a major immunological impact on the infant or young child? This is possible because the developing lung is highly susceptible to immunological and structural changes from inhaled toxicants.^{24–25} Early evidence from a birth cohort study linked early life environmental exposures to allergens, high levels of circulating IgE and early respiratory symptoms to persistent wheezing, lung function deficits and asthma later in life.²⁶ A study based in Los Angeles found that proximity of a subject's home to busy traffic before the age of 2 is associated with increased risk for asthma at ages 5–7 years, but no increased risk was found if exposure occurred after the age of 2.⁴ Other studies have used GIS-based exposure model estimates of traffic-related air pollutants at the birth addresses of infants. These studies found increased traffic exposures were associated with the following: wheeze, doctor-diagnosed asthma, ear/nose/throat infections, flu/serious colds and specific sensitisation to common food allergens during the first four years of life in The Netherlands²⁷; cough without infection and dry cough at night at age 1 year in Munich, Germany²⁸; incident cough without a cold at ages 3–5 years in Leicester, England²⁹; and prevalence of wheezing without a cold in infants living in Cincinnati, Ohio.³⁰ These findings suggest that early in life, traffic exposure may induce allergic sensitisation, acute respiratory infections and asthma-related respiratory symptoms without infection.

For infants and children ages 6–18 diagnosed with asthma, the adverse impact of traffic indices was stronger in females than in males. A stronger effect of traffic-related air pollution in girls has also been observed in other studies examining the relation between traffic-related air pollution and respiratory outcomes.^{4–5–7–9} The sex differences observed are probably attributable to the younger population of boys and girls, who dominate the study population (92.6%) compared with adolescents (7.4%). In addition, the proportion of adolescent subjects with repeat visits was far smaller than younger children (table 1). The underlying reason for the increased susceptibility of younger girls to traffic-related air pollution is not known, but is counter-intuitive given the male structural and immunological predisposition towards airway dysfunction and atopy. Girls have less airway resistance and have larger airways in relation to their lungs than do boys.^{31–32} Girls on average also have higher air flow rates as measured by FEF/FVC (forced expiratory flow/forced vital capacity) and FEV₁/FVC (forced expiratory volume in one second/forced vital capacity) than boys with the same lung size.³³ Low FEF/FVC ratios and small airways relative to lung size have been associated with higher airway sensitivity and reactivity to allergens.³⁴ This type of airway hyper-responsiveness may play a part in the development of asthma. Both genders also differ in their immunological responses to environmental exposures. The rate of atopy as measured by skin

reactivity tests is lower in younger girls than in boys.³³ Consistent with boys' structural and immunological susceptibility, the prevalence of asthma in the United States for young children is consistently higher in boys than in girls.³⁵ Given boys' relative predisposition to respiratory symptoms, it is surprising that females, not males, have an increased risk of repeated hospital encounters with traffic-related exposures. This suggests the propensity towards traffic induced asthma symptoms is not atopic in nature. Interestingly, two studies found that significant associations between proximity to traffic and asthma prevalence were isolated to children with no family history of asthma, which is strongly linked to atopy.^{4–36}

For children 1–5 years old, no significant increase in the rate of repeated hospital encounters was observed. However, asthma is relatively difficult to diagnose in young children, in part owing to the inability to accurately communicate symptoms and complete medical tests, but also to their susceptibility to asthma-like symptoms caused by lower respiratory infections. For young children, wheezing is generally associated with lower respiratory infection and may not be asthma.^{26–37} Physicians presented in hospital with childhood respiratory symptoms such as wheezing may diagnose the condition as asthma without further confirmatory evidence. In one birth cohort study, approximately 60% and 40% of children who wheezed during the second or third year of life, respectively, were transient wheezers, meaning that they stopped wheezing after the age of 3.²⁶ This lack of diagnostic confirmation is a major limitation of the present study and applies to our findings for infants. Children ages 0–5 years who did not have asthma and had a low overall risk may have been misclassified as having asthma, thereby reducing the overall group risk. We have greater confidence in results for children ages 6–18 years because asthma diagnoses are generally much clearer for this age group.

Limitations

We had limited sample size to assess differences in associations between subjects with primary vs. secondary diagnoses of asthma. A sensitivity analysis for the hazard of repeated hospital encounters for children 0–18 years diagnosed with primary asthma showed all HR values >1.0 that were similar to estimates in table 2, but none was statistically significant. The most significant results for traffic density was for subjects in the fourth quintile who had a repeated hospital encounter rate for a primary diagnosis of asthma that was 17% higher than those without major roads within 300 metres of their residences (HR = 1.17; 95% CL 0.95 to 1.44).

The study catchment area is a heavily trafficked urban area with the majority of population residing within 300 metres of major roads. Therefore, limited subject numbers were available for analysis in the referent group and this may have resulted in unstable baseline estimates.

Studies in the area of paediatric respiratory health in recent years have linked genetic factors and environmental exposures to adverse respiratory outcomes.^{38–39} In the present study, we were limited in the number of covariates we were able to incorporate into our analysis owing to reliance on hospital admission data. Consequently, we were not able to assess important covariates that may have confounded the relation between traffic-related air pollution and repeated hospital encounters, including family history of asthma. While it may be assumed that children with a family history of asthma are randomly distributed relative to major roads, it is conceivable that parents with asthma are more sensitive and knowledgeable to asthma triggers and choose to reside further from more urban

settings. If this were the case, the risk of repeated hospital encounters could be biased towards the null hypothesis of no increased risk in association with traffic-related air pollution. Additional environmental exposures noted by previous studies to be associated with atopy, but not assessed in our study, include both indoor and outdoor environmental exposures. These include exposure to background ambient air pollution, secondhand smoke, wood smoke, allergen sources (for example, pests and pets) and number of siblings.^{4–39} This may have led to an overestimation of risk. In the present study, we did not distinguish risk attributable to neighbourhood traffic-related pollution versus regional ambient air pollution, although variability in background air pollution is likely to have been low within the 13-km radius relative to the remaining region of the Los Angeles air basin.

In the present study, we were unable to account for exposure errors because of permanent migration and day migration. We used residential addresses from the first hospital encounter to estimate traffic exposure and assumed minimal permanent migration during the study period. However, this is an assumption we were not able to confirm because there was no longitudinal component that allowed us to collect data on subsequent moves or the duration of residence. We also have no information on day migration for the study cohort. Specifically, there are no corrective data on the time and duration children spent away from their residence in day care, schools or other locations, and we had no data on other pollution exposures at non-residential locations. The study was not designed to fully investigate the effect of a lifetime chronic exposure in relation to acute events. The ideal design would include repeated exposure assessments starting early in life and would also include estimated home exposure data near the times of all admissions to better estimate the impact of acute exposures.

Further limitations of the present exposure assessment pertain to the use of traffic proxies to estimate individual levels of traffic-related air pollution exposures. These proxies only indirectly represent important components of traffic-related air pollution such as polycyclic aromatic hydrocarbons. In addition, we were not able to separate diesel from automobile traffic, which could differ in emission concentrations of potentially important pollutants for different subjects.

We used ADT count information obtained from OCTA to calculate traffic density. Regional databases, such as those obtained from OCTA, may be inaccurate. Insufficient data points, lags in data updates, incomplete geographic coverage and

missing information may have led to some exposure assessment errors.

For a separate study, we obtained admission records for hospitals located in Orange County from the Office of Statewide Health Planning and Development in California. We found 62% of all paediatric asthma admissions in the study's targeted catchment area were seen at UCIMC and CHOC between 2000 and 2002. Therefore, it is likely that some of the children in the current study may have utilised other hospitals for acute asthma-related care or were not given sufficient follow-up time for a readmission to occur. In such cases, the current study's outcome ascertainment is partial and the censoring assumption underlying the analysis is subject to error.

The study was unable to obtain detailed information on each child's asthma history and general health status. Since asthma severity is often greater in older than in younger children, a characterisation of the degree of asthma disease by age group could have helped bring further insights into the effect of age on the relation between traffic exposure and repeated hospital encounters.

CONCLUSIONS

The results of the present study suggest that living within 300 metres of arterial roads or freeways increases the risk for acute respiratory symptoms requiring multiple hospital encounters in children with a primary or secondary diagnosis of asthma. We found associations were stronger in girls than in boys and among subjects without private insurance. Future studies should re-examine gender, age group and socioeconomic differences with a larger sample, include diagnostic confirmation and incorporate the other potentially important covariates discussed. Additional research using this surveillance dataset is planned including more years of follow-up and the use of improved exposure assessment methods that account for pollutant dispersion patterns by prevailing meteorological conditions and other local pollutant sources.⁴⁰

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Competing interests: None.

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Main message

- ▶ Living within 300 metres of arterial roads or freeways increases the risk for acute respiratory symptoms requiring multiple hospital encounters in children with a primary or secondary diagnosis of asthma.
- ▶ Girls with asthma may be more vulnerable to air pollution from traffic.

Policy implication

The results from this study contribute to a growing body of scientific evidence that link traffic-related exposures to adverse respiratory outcomes among children.

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